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## CORN EAR ROTS, STORAGE MOLDS, AND ASSOCIATED HAZARDS

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Ear rots and storage molds occur every year on corn throughout the Midwest. These diseases are serious concerns in corn production because they cause losses in grain yield and quality, and pose potential hazards in harvesting, handling, and feeding grain. Harvesting and handling hazards are generally related to respiratory problems with inhalation of mold spores, while the hazards of feeding moldy grain are generally related to the presence of toxic chemicals (mycotoxins) produced by the fungi. Particularly because of the possibility of mycotoxin production, it is important to recognize ear rots and storage molds and be aware of their toxigenic properties.

The occurrence of ear rots and storage molds is influenced by environmental, and genetic factors, which play a role both in the field and in storage. In addition, production and handling procedures can have positive or detrimental effects on the susceptibility of grain and the development of mold (8). A broad understanding of these factors is necessary to prevent or reduce fungal deterioration of corn.

While fungi are often conveniently classified as ear rot fungi or storage fungi, the distinction is not clear. Those that are considered ear rot fungi, such as *Diplodia maydis* or *Gibberella zeae*, usually develop noticeably in the field, so the majority of damage is done prior to harvest. However, some of these fungi, such as *Fusarium moniliforme*, may infect a large proportion of kernels in the field without causing noticeable symptoms (2). These fungi can continue to develop in storage if conditions are favorable. They typically do less damage in storage because they do not grow at low grain moisture content.

Those fungi that are considered storage molds (*Penicillium*, for example) generally do not cause extensive damage to ears in the field, but some damage may occur. Usually kernels are infected at such low incidence that it is not noticeable, or the spores are contaminating the surface of kernels without having penetrated. The majority of the damage subsequently occurs in storage. These fungi generally can grow at much lower grain moisture content than the ear rot fungi.

### Identification of Ear Rots and Molds

Many of the ear rots and molds can be identified to some extent by their appearance to the naked eye. Characteristics to evaluate are the color and texture of the fungal growth, the



distribution of fungal mycelium on the ear, and field or storage conditions. The following are the most commonly encountered ear and storage fungi in the Midwest.

**Fusarium and Gibberella Ear Rots.** The most common ear diseases through much of the Midwest are caused by fungi in the genus *Fusarium*. This genus contains a number of different species that cause a variety of diseases on many hosts. These fungi have two reproductive stages, each of which is known by a different name. The asexual reproductive stage is encountered most often, and this is the stage known as *Fusarium*. The sexual reproductive stage is less common; most *Fusarium* species found on corn have a sexual stage in the genus *Gibberella*. Other *Fusarium* species have sexual stages in different genera.

On corn, *Fusarium moniliforme* is probably the primary species (4), but the closely related *F. proliferatum*, *F. subglutinans*, and *F. anthophilum* are also common. These fungi all have the same sexual stage, *Gibberella fujikoroi*. *F. graminearum* is also common, but it is usually known by the name of its sexual reproductive stage, *Gibberella zeae*. The common names "Fusarium ear rot" or "Fusarium kernel rot" usually refer to *F. moniliforme*, but can include *F. subglutinans*, *proliferatum*, or *anthophilum*. "Gibberella ear rot", "pink ear rot", or "red ear rot" refers to the sexual stage of *F. graminearum*, *Gibberella zeae*.

Symptoms of Fusarium ear rots are a white to pink or salmon colored mold, beginning anywhere on the ear or scattered throughout. Often the decay begins with insect-damaged kernels, but most of the ear can be affected. Infected kernels are often tan or brown colored, or have white streaks. Fusarium ear rot occurs under a wide range of weather conditions. Fusarium spores are spread by wind and splashing rain to the silks, which are most susceptible for the first 5 days after they appear. The subsequent development of ear rot is heavily influenced by rainfall during the late summer (3). Infection can also occur through the stalk (2), but the importance of this mode of infection has not been established. Insect-damaged kernels are very susceptible, and there is some evidence that insects can act as vectors of the fungus (3). *F. moniliforme* has been reported to grow very little when grain moisture content is below 19% (3). Recently, *F. moniliforme* and *proliferatum* have been shown to produce harmful mycotoxins (see below)(6).

Gibberella ear rot can be identified most readily by the red or pink color of the mold. It almost always begins at the tip of the ear. In some cases, the color is too pale to be seen readily, so the mold appears white. In this case, it may not be possible to distinguish Gibberella ear rot from Fusarium ear rot without a microscope. Gibberella ear rot can be very destructive to the ears, and this fungus produces mycotoxins that are harmful to livestock (see below). The spores of this fungus also infect through silks, but stalk infections and insects are not believed to be important for Gibberella ear rot. Gibberella ear rot infections occur more commonly when the weather is cool and wet during the first 5 days after silking. Continued development of the mold also depends on subsequent cool, wet weather. Optimum temperatures for *F. graminearum* (*Gibberella zeae*) are 65-70 F, but it can grow at much lower temperatures. In storage, *Fusarium* species (including *Gibberella*) appear as white or pink mold, and kernels that appear brown and decayed



**Diplodia ear rot.** Next to the *Fusarium* and *Gibberella* ear rots, *Diplodia* ear rot, caused by *Diplodia maydis*, is probably the most common. This fungus initially appears as a white mold beginning at the base of the ear. The mold and the kernels eventually turn a grayish brown color and rot the entire ear. The mold may be apparent on the outside of the husk. A very distinguishing characteristic of *Diplodia* ear rot is the appearance of raised black bumps on the moldy husk or kernels. These are the pycnidia of the fungus, where new spores are produced. *Diplodia* is spread primarily by splashing water, and infection takes place at the ear shank. Corn borer damage in the shank can provide an entry wound for the pathogen. *Diplodia* ear rot is favored by cool, wet weather during grain fill. Rainfall during August, September and October is correlated with *Diplodia* ear rot incidence (3). *D. maydis* is not known to produce harmful mycotoxins. *D. maydis* appears as a white mold in storage, with kernels appearing brown and decayed. It can be identified by the black pycnidia, if present. Growth of this fungus is very slow when grain moisture is below 21% (3).

*Fusarium*, *Gibberella*, and *Diplodia* also cause stalk rots. They survive in crop debris from year to year. Therefore, continuous corn production favors more severe stalk rot and ear rot. This has been demonstrated for *Diplodia* and *Gibberella*, but may not be as important for the other *Fusarium* species.

**Nigrospora ear rot.** This fungus, *Nigrospora oryzae*, appears as a gray or black mold, often starting at the base of the ear. It produces large black spores, which can be seen with the naked eye. They appear as masses of "pepper" on the surface of the kernels. *Nigrospora* also rots the cob, giving it a brown, tattered appearance at the shank.

**Cladosporium ear rot.** *Cladosporium herbarum* and other species often infect damaged kernels. This fungus appears gray to black or very dark green, and can have a powdery appearance. It also causes black streaks in the kernels. This disease can be fairly common but usually does not cause extensive damage to the ears. In storage, it can be identified by the black powdery spores and black kernel streaks. *Cladosporium* and *Nigrospora* are not known to produce harmful mycotoxins.

**Aspergillus ear rot and storage mold.** *Aspergillus flavus* and *A. parasiticus* are generally known as storage fungi, but they can also cause ear rots in the field. *Aspergillus* is a gray-green, powdery mold. In Iowa, it is much more common in hot, dry years. It can grow at temperatures higher than 90 F, and grain moisture content as low as 16% (3). These fungi produce the most well-known mycotoxins in corn, aflatoxins. The fungus can be detected in corn because it is fluorescent under black light, but this does not indicate the presence of aflatoxins.

**Penicillium molds.** Several species of *Penicillium* attack corn kernels, primarily in storage. Like *Aspergillus*, this fungus can also cause ear damage in the field. *Penicillium* is a blue-green, powdery mold. It most closely resembles *Cladosporium*, but it can sometimes be confused with *Aspergillus*. These three can be distinguished by their color, and by their spores under a



microscope. Some *Penicillium* species can grow at grain moisture contents of 16-17% (3). Kernels with "blue eye" have embryos infected with *Penicillium*. Generally, *Penicillium* does not cause mycotoxin problems, but certain *Penicillium* species can produce them.

### Prevalence of Ear Rots

Surveys of ear rots have been conducted in Indiana and a similar survey is underway in Iowa. The Indiana study reported data on four major ear rots: *Fusarium*, *Gibberella*, *Diplodia*, and *Aspergillus* (Table 1). The results of the Iowa survey are not yet complete, but *Fusarium* ear rots (*F. moniliforme*, *proliferatum*, *subglutinans*) are the most common (92% of fields), followed by *Cladosporium* (51%), *Gibberella* (42%), and *Nigrospora* ear rots (9%). *Diplodia* ear rot has been less common in the survey, but severe cases have been observed this year in some fields.

### Mycotoxins

Mycotoxins are the major hazard associated with ear rots and storage molds. While many of these toxins are believed to be harmful to humans, hazards to livestock are the primary concern. A wide variety of toxins can be produced by many different fungi, but for this discussion, the major toxins fall into two groups: those produced by *Fusarium* species (vomitoxin, zearalenone, T-2 toxin, fumonisins, and others) and those produced by *Aspergillus* species (aflatoxins). Some *Penicillium* species can produce toxins, but they will not be discussed here.

Within the genus *Fusarium*, the major toxigenic species on corn are *F. graminearum* (*Gibberella zeae*), *F. moniliforme*, and *F. proliferatum*. Other toxigenic species on corn include *F. subglutinans*, *F. anthophilum*, *F. poae*, *F. sporotrichioides*, and several others (5). Although each species may be capable of producing a range of toxins, particular toxins are more strongly associated with particular species. I will not attempt to list all the toxins or toxigenic species here. The following is a discussion of the more common and important toxins found in Iowa corn.

Deoxynivalenol (DON), or vomitoxin, is one of the trichothecene toxins. This toxin is probably the most commonly associated toxin with Iowa corn. Trichothecenes are a large family of mycotoxins produced by several *Fusarium* species, notably *F. graminearum*. Generally, these toxins cause reduced feed intake, weight loss, vomiting, bloody diarrhea, suppressed immune system, hemorrhage, abortion, and death (1). Vomitoxin causes reduced feed intake in swine at levels above 1 ppm. At 10 ppm or more, complete feed refusal or vomiting can occur. Higher levels can cause some of the more severe symptoms described above. The Food and Drug Administration recommends that for swine, DON levels in feed should be below 1 ppm. For cattle, the FDA recommends levels below 5 ppm. Another trichothecene, T-2 toxin, has sometimes been detected in Iowa corn.

Zearalenone is another toxin produced by *F. graminearum*, and commonly occurring in combination with vomitoxin. The effects of zearalenone are generally related to livestock



reproduction. These effects include reduced litter size, vaginal prolapse, atrophy of ovaries or testicles, embryonic death, and abortion. These effects can occur at levels as low as 1 ppm in young swine. Cattle are less susceptible, and effects are not usually seen at levels below 10 ppm.

Fumonisin is a group of toxins produced by *Fusarium moniliforme* and *F. proliferatum*. This is a more recently recognized problem. Although *F. moniliforme* has been associated with animal health problems for many years, and is known to produce other mycotoxins (6), the cause of some of these health problems was unknown until 1988 (6). These toxins were discovered in South Africa as a result of research on the cause of high levels of esophageal cancer in humans in southern Africa. Fumonisin is associated with the increased cancer levels. In livestock, the primary diseases caused by fumonisin are leukoencephalomalacia in horses and pulmonary edema in swine. Both are fatal. The disease in horses is a degeneration of the brain that results in staggering, blindness, delirium, and death. In swine, the disease is a degeneration of the lung tissue. In 1989, outbreaks of both diseases occurred in the U.S., and this led to the first direct evidence that fumonisin caused these diseases. In most cases, animals had been fed corn screenings which were found to contain up to 330 ppm fumonisin B1. Although more data are needed, it appears that fumonisin concentration of about 10 ppm are sufficient to induce either of these diseases.

Aflatoxins are probably the most well-known mycotoxins in corn. These toxins are produced by *Aspergillus flavus* and *A. parasiticus*. While these are among the most potent mycotoxins, their occurrence in the Midwest is sporadic, and they do not pose a consistent threat. One reason for heightened concern over aflatoxin is that acceptable levels are regulated by the federal government. The primary effects of aflatoxin are potentially fatal liver and kidney damage or cancer. Guidelines for aflatoxins are as follows: milk, <0.5 ppb; corn for lactating dairy cows, young animals, or for unknown use, <20 ppb; corn for breeding cattle, swine, mature poultry, <100 ppb; finishing swine, <200 ppb; finishing cattle, <300 ppb.

### Testing for Toxins

Corn or feed should be tested for mycotoxins if symptoms are noticed in animals that have consumed the feed, or if there is evidence of extensive mold damage by a toxigenic species. Producers should check for mold problems in the field so decisions can be made before the corn has been harvested. Grain in storage or coming out of storage should also be checked for evidence of mold. Several of the molds do not produce toxins, so a mold problem does not always indicate a toxin problem. Once a toxigenic mold has been identified, this is a warning sign, but a toxin problem still does not necessarily exist. The use of blacklight to detect *Aspergillus flavus* does not directly detect the toxin, so this should be used only as a warning sign. Toxigenic fungi do not produce toxins under all conditions. Toxin detection is the only certain way to determine if a problem exists.



Mycotoxins can be detected by immunological methods or by analytical chemistry methods. The chemistry of mycotoxin analysis will not be discussed here. Detection of toxins in the field or at the elevator can be accomplished through the use of several available kits that usually use immunological methods for toxin detection. These results are usually qualitative or semiquantitative. Quantitative results require the use of chromatography. This type of analysis can be obtained through the services of commercial or state-run laboratories.

Sampling is very important when submitting a specimen for mycotoxin testing. A representative sample should be taken from several locations in the field, bin, or truck. A general rule is to take about a 1 lb. sample for every ton of grain (1). The samples should then be thoroughly blended and the entire sample ground together prior to analysis.

### **Harvest and Handling Hazards**

Harvesting and handling grain can be hazardous, and the presence of molds in grain can increase some of these hazards. A more thorough discussion of safety in grain handling can be found elsewhere in these proceedings, but I will focus on those aspects involving molds.

Respiratory hazards are the primary human risk when harvesting and handling moldy grain. Inhalation of grain dust can cause respiratory problems, and high levels of mold spores can increase the amount of dust and the amount of respiratory difficulties. Sensitivity to mold spores is not necessarily related to toxigenic properties of the fungi. Long-term exposure to dust and spores can have serious effects, and it pays to be aware that the hazards are real. Protection from dust inhalation can be achieved through the use of a paper mask, although this provides minimum protection. A respirator provides much better protection, while sensitive individuals working in a very dusty environment might require a helmet with an air supply.

Another hazard of working with moldy grain is related to safety in the grain bin. Moldy grain often clumps together and causes problems in moving grain. Accidents can happen when you enter a grain bin to break up these clumps.

### **Preventing Molds and Toxins**

The prevention of ear rots is a difficult task, but there are some strategies for reducing their occurrence. Most of the fungi causing ear rots survive in crop debris. Insect damage is closely linked to incidence of Fusarium ear rots, so insect control can be of some value. Reducing the amount of debris through crop rotation or tillage can reduce the risk of disease, especially for Diplodia ear rot. An early harvest can prevent the development of ear rots. If extensive ear rot development is observed, a field should be harvested as soon as moisture content reaches a level that can be combined. Some hybrids are more resistant to ear rots than others, but overall, resistance to ear rots is not widely available. Some ear and kernel characteristics favor less ear rot, but these may have negative side effects. For example, kernels with a thick pericarp are more



resistant to infection, but also dry very slowly. In general, hybrids with tight husks and ears that do not remain upright experience less ear rot.

Damage that occurs during harvest can lead to subsequent mold development. Properly adjusted equipment can reduce the amount of damage (8). Cleaning grain before drying will remove much of the particles and damaged kernels. These are often the moldiest and most toxic component of harvested grain.

After harvest, moldy corn should be dried immediately to 15% moisture or less. Holding grain for even a short time can allow significant mold development. For long term storage, 13-14% moisture is recommended. This treatment will minimize mold growth, but may have certain disadvantages. Slower drying causes less drying damage and is less expensive, but may allow the development of molds. The aggressiveness of a drying program should depend on the extent of the mold problem. Keep in mind that even clean looking grain will develop mold problems if not dried and stored properly. Bins should be thoroughly cleaned before the new crop is stored. Aeration and stirring will help prevent pockets of mold. Grain in storage should be checked periodically. Antifungal agents can be applied to grain to reduce mold growth in storage. These products, such as propionic acid, do not kill the mold already present or reduce toxins already formed. They may have other disadvantages, such as restricting utilization of the corn.

### Conclusions

Ear rots and storage molds are a consistent problem, causing reductions in grain yield and quality. Weather conditions beyond our control will largely dictate the severity of ear rot problems. There are no quick solutions to the occurrence of these diseases, but scouting fields and handling grain properly will keep problems to a minimum. Toxin problems can and do occur, but moldy grain is not always toxic, even when toxigenic fungal species are involved. Genetic resistance to infection and toxin production may eventually reduce the seriousness of ear rots and storage molds.

Table 1. Incidence of ear rots in Indiana in 1989-1991 (7).

	1989	1990	1991
Fields with ear rots (%)	91.7	68.1	98.8
<i>Fusarium</i>	89.9	56.4	96.4
<i>Gibberella</i>	14.2	28.8	9.6
<i>Diplodia</i>	3.6	6.1	2.4
<i>Aspergillus</i>	1.2	0.0	4.2
Others	47.9	12.9	42.3



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